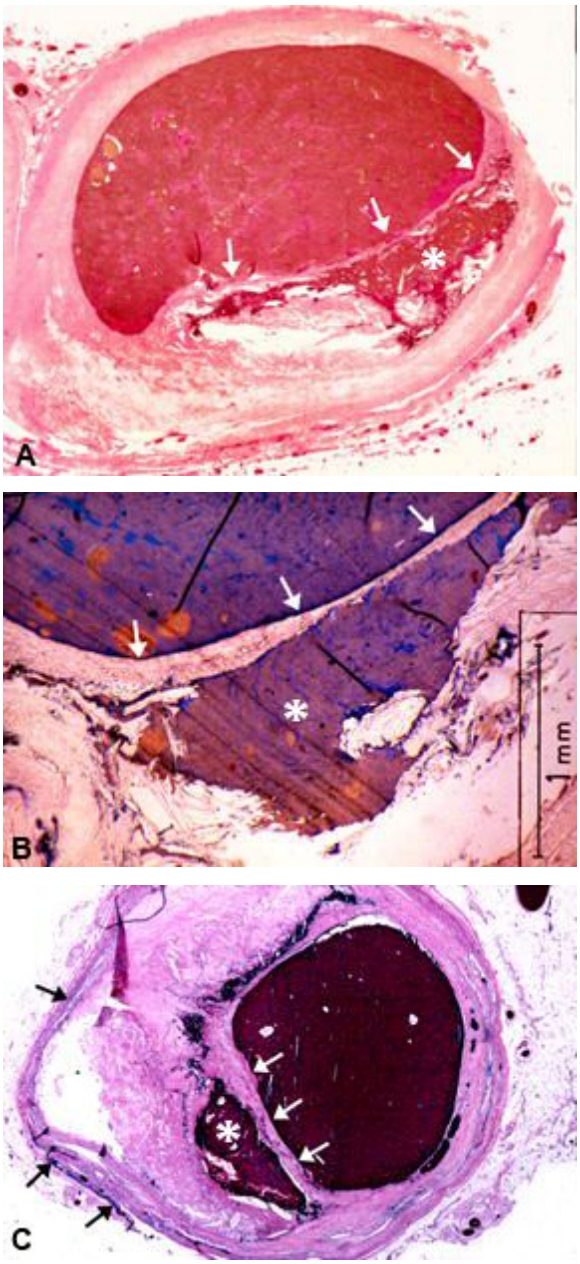
8. Blind Pockets and False Channels

"An unusual variation of this phenomenon was found in even fewer cases where rupture and subsequent extrusion of plaque contents occurred, leaving only a shelled out mural plaque."
RL Ridolfi, et al., [67]

We have previously emphasized (Chapter 4) that the growth of the necrotic core is associated with increased intracore pressure leading to spontaneous ulceration and drainage of the core itself [66,117,118]. Spontaneous ulceration represents the culmination of factors and forces internal or external to a necrotic core, resulting in the removal or partial removal of necrotic material from the artery wall [4]. This chapter will further explore the mechanism of PU and show that some plaques may drain by forming false channels that course through the body of the necrotic core. It will also consider the role of blind pockets, created by the discharge of plaque contents, in the pathogenesis of acute coronary occlusion.

False Channels

Figure 18 illustrates replacement of necrotic core contents by injection mass in five different patients, reflecting pre-mortem ulceration and drainage of the necrotic core. None of these UPs was associated with an occlusive thrombosis, nor did any of these patients receive thrombolytic drugs. The tissue surrounding the margins of these empty atheromas have red blood cells, providing further evidence of pre-mortem formation. The fibrous cap overlying the empty core is intact, suggesting these empty plaques are, in reality, false channels. Histologic examination, both proximal and distal, showed these false channels extended anywhere from 4-to-25mm in length. The formation of a false channel requires both a proximal entrance point and a distal exit, and the flow of blood through this channel results in the emptying or "wash out" of plaque contents into the distal circulation. The end result is a "shelled out" appearance to the necrotic core, as shown in Figure 18, reducing plaque bulk and, possibly luminal stenosis also [67].



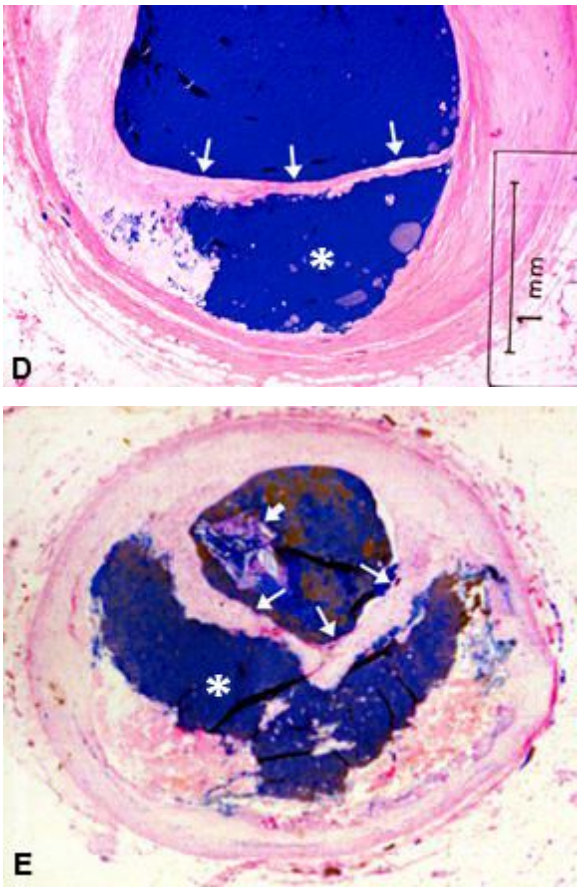


Figure 18: A - E. These figures show empty, "shelled-out," atheromas in 5 different patients. The fibrous cap (thin white arrows) is intact in all figures, but plaque contents are replaced by colored injection mass. White asterisk = false lumen. Significant stenosis of the true lumen is present in **C** and **E**. Thrombus is absent in both true and false lumen in all figures. An embolic fragment of plaque tissue (fat white arrow) is present in the lumen in **E**. Adventitial inflammation (black arrows) is present in **C**.

Formation of False Channels

Chapters 1 and 5 pointed out that plaques grow both circumferentially and longitudinally, and that adjacent, but separate, plaques often extend and fuse together to form larger and longer plaques. A long plaque with a long necrotic core will have an extensive shoulder area, extending down both sides of the plaque. Since PU commonly occurs at the shoulder of the plaque, the possibility exists that a long necrotic core may develop one or multiple shoulder ulcerations along its length. Blood entering the necrotic core at a proximal site of ulceration may then exit through a distal ulcerated site without disrupting the overlying fibrous cap, creating a false channel [119].

Figure 19 is an example of a false channel involving a long segment of the mid-LAD coronary artery in a 58-year-old man who died of inferior wall cardiac rupture several hours after receiving streptokinase for an acute right coronary thrombotic occlusion. The fibrous cap is beginning to disintegrate in the mid-portion of the plaque, Figures 19C and 19D, but the cap is still intact proximally, Figure 19B, and distally, Figure 19E. The core contents are completely gone from this plaque except for some residual remnants at the distal end of the false channel, Figure 19E. Significant luminal stenosis is absent in the area of ulceration, and there is no evidence of thrombus formation.

We hypothesize that before ulceration and drainage, this was a relatively large plaque with a long necrotic core that subsequently drained and debulked without human intervention. Presumably, with core contents removed, the area will resolve, stabilize, and reendothelialize, resulting in an overall reduction in luminal stenosis [68,120]. The absence of thrombus suggests that resolution and reendothelialization take place by in-growth of surrounding endothelium, probably over a bed of platelets. The process of resolution of an UP is probably aided by complete or relatively complete drainage of plaque contents from the area, similar to a bacterial abscess.

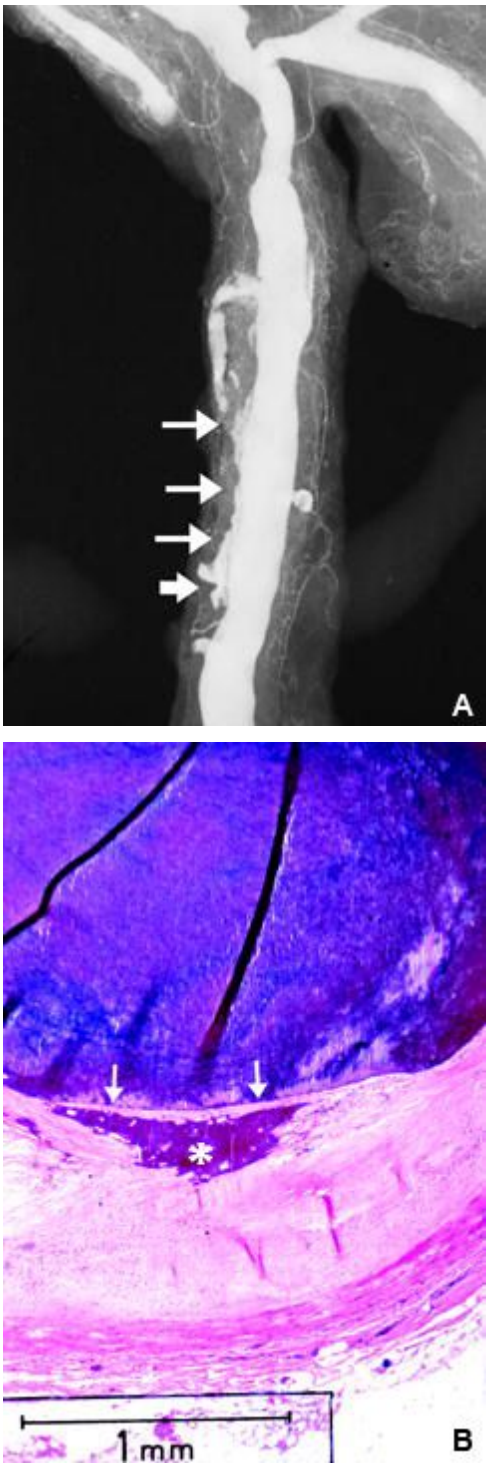


Figure 19: **A**, Post-mortem angiogram showing extensive ulceration (white arrows) without luminal stenosis of the proximal LAD coronary artery of a 58-year-old white male. A blind pocket is present at the distal end of the UP (fat white arrow). **B-E**, Contiguous coronary sections taken proximal (**B**) to distal (**E**) from the area of ulceration. The ulceration is approximately 25 to 30mm in length. These sections show the presence of a large false channel (white asterisk) in which the fibrous cap (white arrows) overlying the false channel has become thin in **B**, begins to disintegrate in **C** and **D**, and then is again intact in **E**. Note the absence of thrombus in all figures and presence of residual plaque contents (black arrows) in **E**. Reproduced with permission [see Ref 57].

Frequency of False Channels

How often do false channels develop in the course of active progressive atherosclerotic disease? Is the development of false channels a rare phenomenon, or a common occurrence with ulcerating atheromas? Are spontaneous coronary dissections or coronary dissections following PTCA in reality false channels coursing through a necrotic core along cleavage planes, as illustrated and discussed in Chapter 4? A previous report from this laboratory showed the longitudinal extent of 109 UPs without associated luminal thrombosis [57]. Fifty-five percent (55%) of these UPs involved two or more contiguous segments and 32% involved three or more contiguous segments. Since the coronary arteries were cut at 2-3mm intervals, this means that 1/3 of these UPs were, at a minimum, 6-to-9 mm in length. It is relatively common for UPs to extend long distances within the artery wall, with the potential to develop a false channel. We hypothesize that all UPs have the potential to extend longitudinally and to develop false channels if the underlying pathologic substrate is suitable or favorable for their formation.

Blind Pockets

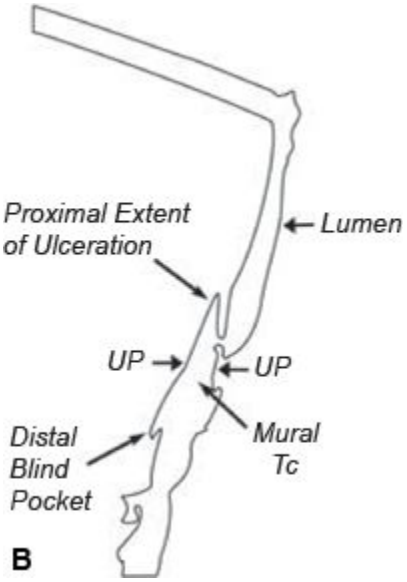
The majority of plaques that ulcerate do not go on to develop false channels. It is our theory that this is due to anatomic and structural features or to “geometric” changes within and adjacent to the ulcerated plaque [121]. The formation of a false channel depends upon the development of a distal exit point, and failure to develop a false channel creates what is essentially a “blind pocket” or cavity in the wall of the artery. Blind pockets form in association with UPs when there are obstructions within the necrotic core, particularly at the distal end, and/or the surrounding arterial wall, that prevent the formation of a distal exit point (Figure 19E). These obstructions include the presence of fibrous septa criss crossing or dividing the necrotic core, a very thick and/or strong fibrous cap covering the distal extent of the core, calcification of intimal tissue distal to the core, and the presence of an arterial bifurcation. These anatomic features will be different in every patient and every plaque and may help to explain the great variation among UPs and their associated complications.

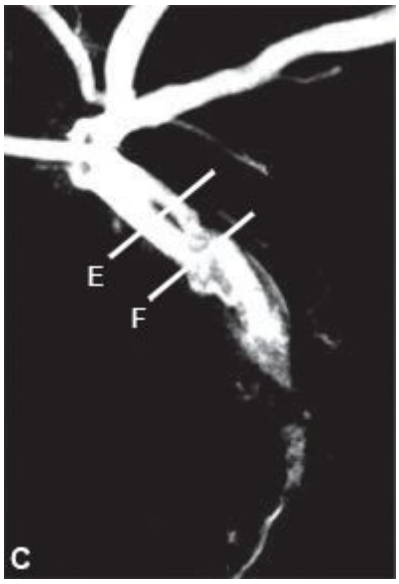
Blind Pockets and Acute Occlusions

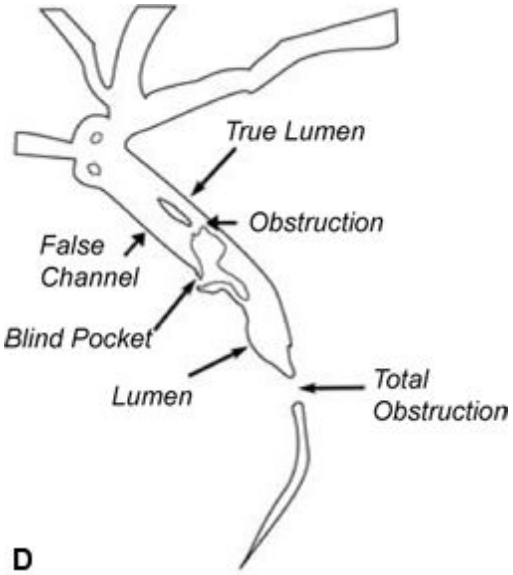
Figure 20 illustrates examples of blind pockets in two patients who died of ACD. In Figures 20A and 20B an UP is associated with both a proximal and distal blind pocket. A mural thrombus is located at the site of the UP and a remnant of fibrous cap is mixed with the thrombus. The patient received streptokinase to treat an acute anterior myocardial infarction, thought to be caused by this lesion, but died shortly thereafter of anterior wall rupture. This patient illustrates the association between a blind pocket, dislodgement of the fibrous cap, and formation of an occlusive thrombus. Dislodging the moorings of the fibrous cap, especially that portion of the cap covering the distal end of an UP, may lead to acute occlusion of the lumen, caused by a flap of fibrous cap. The thrombus in this patient was partially lysed by the streptokinase, but the pathologic substrate, consisting of a blind pocket and a flap of fibrous cap, were not changed by this clot lysis.

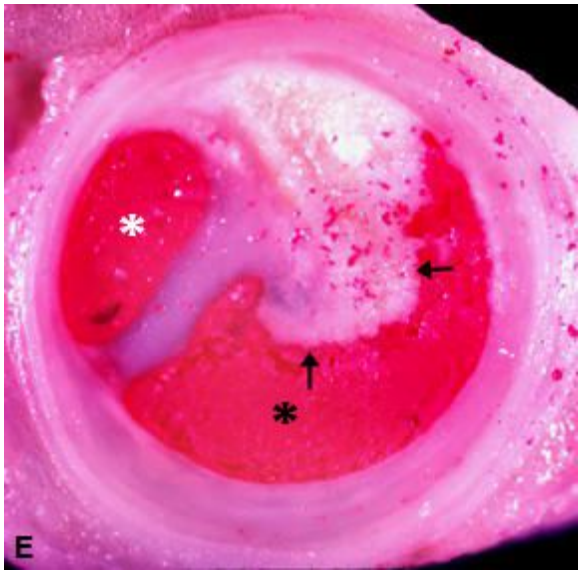
Figures 20C-F, shows severe luminal stenosis produced by swelling and expansion of a blind pocket at the distal end of the false channel. This example illustrates how sudden swelling of a blind pocket can lead to rapid and severe luminal stenosis, without the formation of an acute thrombus. It is easy to visualize how the pulsating head of pressure that drives coronary blood flow could act much like a battering ram, not only in distending the core area, but also in dislodging the fibrous cap. Acutely

developing luminal stenosis, caused by either sudden swelling of the plaque or by a flap of fibrous cap, could precipitate acute coronary events in the absence of occlusive thrombosis.









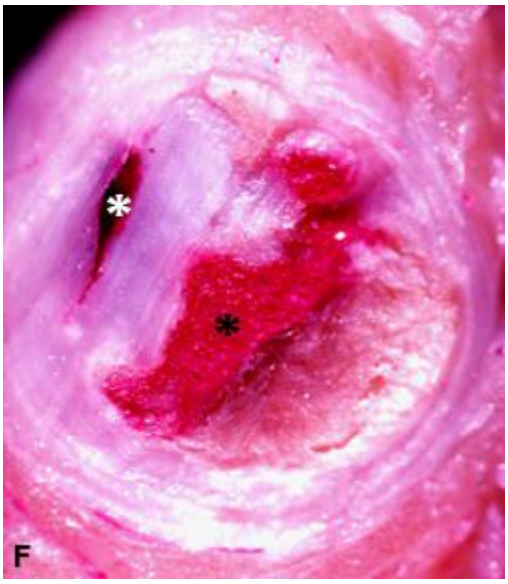


Figure 20: Post-mortem angiograms of dissected LAD coronary arteries of a 71-year-old male (**A**) and a 62-year-old male (**C**). **B** & **D** are schematic diagrams of the X-rays in **A** & **C**. UP = ulcerated plaque. Tc = thrombus. In **A**, the ulceration involves the central part of a long necrotic core, with injection mass filling the proximal portion of the atheroma and also a distal blind pocket. A mural thrombus partially occludes the lumen at the site of the ulceration and includes a remnant of the fibrous cap (not shown). **E** & **F** show both a true lumen (white asterisk) and a much larger false lumen (black asterisk), with the false lumen ending in a blind pocket in the patient shown in **C**. A small mural thrombus is present (black arrows) in **E**.The true lumen is reduced to a tiny slit, apparently the result of distention and swelling of the necrotic core. The true lumen and the false lumen both contain thrombus distal to **E** (not shown).

Clinical Implications

Some acute coronary occlusions respond to thrombolytic drugs, but others do not [122]. If all acute coronary occlusions were caused by pure thrombus, then we would expect 100% of thrombi to be lysed by a thrombolytic drug. But there are different types and kinds of acute coronary occlusions, and one treatment is not suitable for all. A thrombus formed proximal to an occluding fibrous cap may not lyse, with blood flow restored, because the fibrous cap does not respond to a thrombolytic drug.

However, penetration of the obstruction, presumably a fibrous cap flap, with a guide wire allows the thrombolytic drug to perfuse down the artery and to lyse the thrombus [123]. Similarly, acute occlusion caused by distention and swelling of the plaque, as in Figures 20C-F, will not respond to a thrombolytic drug because the occlusion is not caused by a thrombus. This may explain why PTCA is more successful than thrombolytic drugs to treat acute coronary occlusion [124]. For example, balloon dilatation of an obstruction caused by a flap of fibrous cap or a distended blind pocket, followed by stent placement, will serve, first, to open the channel by breaking up any fibrous obstruction, and, second will produce closure of blind pockets, tacking up loose ends of fibrous cap [125]. This approach tends to correct the pathologic substrate of a blind pocket and a flap of fibrous cap, and to close any potential dissection planes that were responsible for the occlusion in the first place [126]. Stent place ment may also be beneficial in producing more complete drainage of the necrotic core through squeezing or compressing the plaque against the artery wall. Complete drainage of the necrotic core through the use of a stent tends to stabilize the plaque, facilitate resolution and healing, and, in the end, to reduce luminal stenosis. We hypothesize that the pathogenesis of many occlusive thrombi associated with an UP develop when blood cannot exit the necrotic core, or, in essence, are due to the presence of a blind pocket and the failure to develop a false channel.

In Review

Spontaneous ulceration and discharge of an atheroma may result in a diverticulum-like structure in the artery wall. It contains a mixture of plaque contents and blood in what is essentially a blind pocket. Over-distention of this blind pocket by inflowing blood can lead to increased luminal stenosis and obstruction to coronary flow, before throm-

bus formation. Pulsatile blood flow, acting like a battering ram within the blind pocket, may promote the formation of a distal point of reentry, creating a false channel within the artery wall. Formation of a false channel with relatively complete drainage of core contents results in a reduction in luminal stenosis without human intervention. The formation of a false channel is another mechanism by which toxic plaque contents are removed from the wall, and it may occur without producing occlusive thrombosis or acute coronary events. All UPs have the potential to develop a false channel. The presence of a blind pocket, associated with the failure to develop a false channel, constitutes a common pathologic substrate that underlies the development of acute coronary events. Obstruction of coronary flow by a fibrous cap flap is one explanation for the failure of thrombolytic drugs in patients with acute S-T segment elevation myocardial infarction.